Dissertation on

A CLINICAL STUDY ON THE OUTCOME OF TENOPLASTY IN OCULAR CHEMICAL INJURIES

Submitted in partial fulfilment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI- 600 003

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2013

CERTIFICATE

This is to certify that this dissertation entitled **" A CLINICAL STUDY ON THE OUTCOME OF TENOPLASTY IN OCULAR CHEMICAL INJURIES."** is a bonafide record of the research work done by **Dr. R. NAVEEN.,** Post graduate in Regional Institute of Ophthalmology, Madras Medical College and, Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2010-2013.

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
Dr. Naveen .R
PG in MS Ophthalmology
Madras Medical College, Chennai -3

Dear Dr. Naveen .R

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " A clinical study on the outcome of tenoplasty on limbal vasculature in post – chemical injury limbal ischemia " No. 09112011

The following members of Ethics Committee were present in the meeting held on 22.11.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan. MD	-- Chairperson
2.	Prof. A. Sundaram MD Vice principal, Madras Medical College, Ch -3	-- Member Secretary
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8.	Thiru. S. Govindsamy. BA BL	-- Lawyer
9.	Tmt. Arnold soulina MA	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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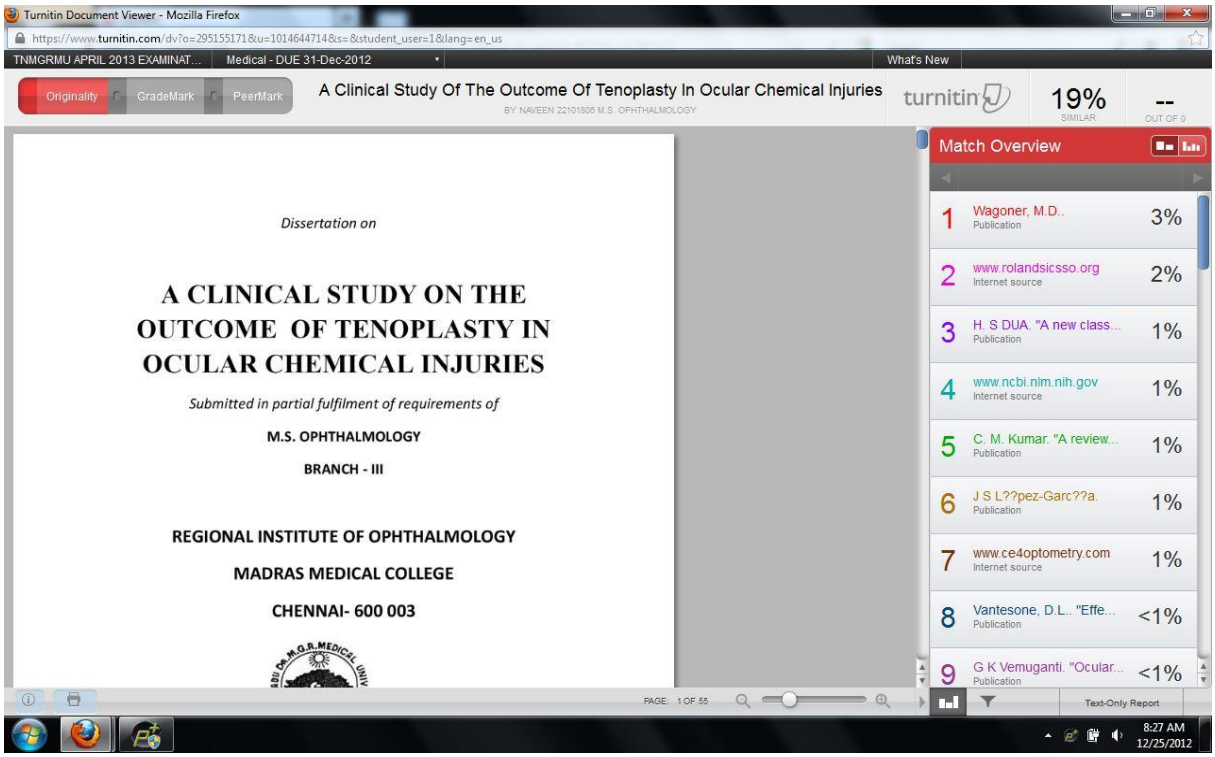
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A Clinical Study Of The Outcome Of Tenoplasty In Ocular Chemical Injuries
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PART - I

INTRODUCTION

CHEMICAL INJURY OF THE EYE

Chemical injuries are devastating ocular surface injuries which can lead to permanent visual loss. It is mainly by industrial accidents, criminal assaults, accidents at home. The most important agents causing are alkali and acid injury. Alkali injuries are more common than acid injuries[1]. Lime is the most common alkali injury. Ammonia causes the most severe damage. Other alkali are lye, potassium hydroxide and magnesium hydroxide. Among acids, sulphuric acid is the most commonly encountered acid injury. The most severe acid injury is with Hydrofluoric acid[2]. Other acids commonly causing chemical injury are sulphurous, nitrous, acetic, chromic and hydrochloric acid.

ANATOMY

CORNEA

The adult human cornea is about 11-12 mm horizontally 9-11 mm vertically. Its approximately 0.5mm thick in the centre and 0.75mm in periphery. It consists of five layers namely corneal epithelium ,Bowman's membrane, stroma, descemets membrane and endothelium. Corneal epithelium is continous with the conjunctival epithelium which is composed of non-keratinised stratified squamous epithelium .Only the basal cells of the corneal epithelium proliferate. Bowmans layer is an acellular membrane like zone at the interface between corneal epithelium and stroma. Corneal stroma forms more than 90% of the corneal thickness and is responsible for cornea physical strength and transparency and stability. There is uniform arrangement of collagen fibres in the stroma which is essential for its transparency. Stroma also contains spindle shaped keratocytes which are similar to fibroblasts .Collagen in corneal stroma is mostly type 1.Descemets membrane is the basement membrane of corneal endothelium. It is mainly made of type IV and type VIII. A single layer of corneal endothelial cells covers the posterior surface of descemets membrane in a well arranged mosaic pattern. The cell density is about 3500 cells/sq mm. It constantly decreases with age[3]. Its main function is to keep the cornea dehydrated and thereby maintaining the transparency.

LIMBUS

It is the corneoscleral junction measuring about 1.5 to 2.0 mm wide. At the limbus, the corneal epithelium becomes the bulbar conjunctival epithelium. the epithelium in this transition zone contains basal cells which are needed for the regeneration of the corneal epithelium. The epithelium is thrown into folds at this transition zone which increases the surface area. These folds are known as the Pallisades of Vogt. The connective tissue within the folds contain blood vessels and lymphatics. The Bowman's membrane of cornea becomes continuous with lamina propria of the conjunctiva and the fascial sheath of the eyeball, the Tenon's capsule. The corneal stroma becomes the sclera. Descemet's membrane ends abruptly at schwalbe's line. The corneal endothelium is continuous with the endothelium of trabecular meshwork

TENON'S CAPSULE

It is a thin fascial membrane that envelops the eyeball and separates it from the orbital fat. The fascial sheath is firmly attached anteriorly to the sclera about 1.5mm posterior to the corneoscleral junction. It fuses with the meninges around the optic nerve. The ciliary nerves and vessels pierces the fascia close to the optic nerve.

PATHOPHYSIOLOGY

PATHOPHYSIOLOGY OF CHEMICAL INJURY

The severity of chemical injury depends on the surface area of contact and the degree of penetration[4]. Damage occurs to the conjunctival and corneal epithelium, the limbal stem cells, stromal keratocytes, stromal nerve endings, endothelium, iris, lens, ciliary body and vascular endothelium of the conjunctiva. Acid injuries are more superficial than alkali injuries[5].

The major principles guiding the evaluation and treatment of chemical injuries are

1. regeneration of ocular surface epithelium,
2. stromal matrix remodelling
3. inflammation

Epithelial Injury, Repair And Differentiation:

The centripetal movement of cells from the peripheral cornea , limbus and conjunctiva is responsible for normal and posttraumatic replacement of corneal epithelium[6]. The presence of limbal stem cells maintains and replaces injured corneal epithelium. The migrated epithelial cells from the conjunctival epithelium never fully expresses corneal phenotypic feature[7]. Reliance on the conjunctival epithelium to resurface the cornea leads to

delayed rehabilitation, deep stromal vascularisation, persistence of globlet cells in cornea and poor epithelial basement membrane adhesion.

Reestablishment of phenotypically normal corneal epithelial surface with limbal stem cell derived cell population is the major principle of treatment of severe chemical injury.

Corneal stromal matrix injury, repair & ulceration:

The primary response of keratocytes is to maintain & regeneration of stroma. After chemical injury, degeneration of the basement membrane collagen and corneal stromal is initiated by various matrix metalloproteases and leads to sterile corneal ulceration[8].

Inflammation :

The infiltration of polymorphonuclea leukocytes into the corneal stroma with sterile corneal ulceration causing inflammation and delay in re-epithelisation and perpetuate continued recruitment of additional inflammatory cells. Rigorous control of inflammation is one of the major principles of chemical injury management.

CLINICAL COURSE &

EVALUATION

CLINICAL COURSE & EVALUATION

There are four distinct pathological and clinical phases in the course of chemical injury:

1. Immediate phase
2. Acute (0-7 days)
3. Early repair (7-21 days)
4. Late repair (21days to several months)

Immediate Phase :

Immediately after the chemical injury, the clinical features are related to the depth of penetration and the relative toxicity and concentration of the injurious compound. The size of corneal & conjunctival epithelial defects determines the extent and the depth of intraocular penetration and it is estimated by corneal clarity, intraocular inflammation, intraocular presence & lens clarity. Limbal stem cell damage is estimated by vascular ischemia and necrosis of limbal & bulbar conjunctiva.

Acute Phase :

The important parameters to be monitored in the 1st week are, evidence of reepithelisation, intraocular pressure and progressive ocular inflammation. Reepithelisation is mild to moderate chemical injuries but absent in severe chemical injury. Intraocular pressure may raise due to trabecular meshwork distortion and release of prostaglandins and may remain elevated due to persistent inflammation.

Early repair phase :

The epithelial migration occurs in early repair phase in mild and moderate injuries but is delayed in severe chemical injuries. In severe chemical injuries a second wave of infiltration of inflammatory cells begin after 7 days and continues to progress over the next several weeks and there is persistence of corneal & conjunctival epithelial defects and limbal ischemia.

Late Phase :

When the late repair phase begins, corneal inflammation, collagen synthesis and collagenase activity are peaking[9]. The balance of wound repair and collagenolysis is greatly influenced by the status of the corneal

epithelium. in mild limbal injury there is normal re-epithelisation of the cornea. In patients with moderate limbal stem cell loss, there is persistence of epithelial defect corresponding to the quadrant of limbal stem cell loss. Further it may lead to re-epithelisation with conjunctival epithelial cells which is associated with superficial pannus. The absence of re-epithelisation in this stage indicates complete limbal stem cell loss. If the proximal conjunctiva is normal, then the cornea heals with total vascularised pannus. In patients with complete loss of limbal stem cell loss and the promimal conjunctival epithelium with ischemic necrosis, there occurs sterile corneal ulceration and perforation.

GRADING OF CHEMICAL INJURY

DUA's CLASSIFICATION OF CHEMICAL INJURY

Grade	Clinical findings	Conjunctival involvement	Prognosis
I	0 clock hours of limbal involvement	0%	Very good
II	<3 clock hours of limbal involvement	<30%	Good
III	>3–6 clock hours of limbal involvement	>30–50%	Good
IV	>6–9 clock hours of limbal involvement	>50–75%	Good to guarded
V	>9–<12 clock hours of limbal involvement	>75–<100%	Guarded to poor
VI	Total limbus (12 clock hours) involved	Total conjunctiva (100%) involved	Very poor

TREATMENT

TREATMENT

IMMEDIATE TREATMENT

Neutralisation of the pH and removal of the remaining debris by copious irrigation with any non toxic irrigating solution for a minimum of 30 minutes prevents further damage to the ocular surface. Debridement of the necrotic corneal epithelium is needed for proper re-epithelisation and healing. The limbal stem cells are placed in the deep location, and hence prompt removal of the debris can prevent permanent damage[10]. Double eversion of the upper eyelid and removal of the debris deposited in the upper tarsal conjunctiva should be done to prevent continued slow release of the chemical into the tear film. Debridement of the necrotic conjunctiva should be done as its a source of inflammatory cells and proteolytic enzymes[11].

Medical therapy

Promotion of epithelial wound healing

The rate-limiting factor in the healing of the chemical injury is the recovery of intact normal epithelium. Re-epithelisation is facilitated by

initial aggressive medical management. In severe injuries surgical treatment should be planned as medical management alone will not be adequate.

Tear substitutes:

In chemical injury, unpreserved tear substitutes are used to facilitate reepithelialization. Topical viscoelastics are used to facilitate reepithelialization in severe chemical injuries. It facilitates corneal epithelial migration in grade I and II injuries and minimizes conjunctival scarring and after grade III and IV injuries it prevents symblepharon formation.

Frequent application of unpreserved tear substitutes and ointments at bedtime after reepithelialization is necessary to prevent recurrent epithelial erosions and persistent keratopathy .

Occclusive Therapy:

Patching of the eyelids protects the migrating epithelial cells in the immediate phase. The cause of epithelial defects in the early and late repair phases is persistent inflammation and limbal stem-cell deficiency which are unresponsive to occlusive therapy.

Bandage Soft contact lens:

Therapeutic soft contact lenses and collagen shields are poorly tolerated in the acute phase and there is no advantage to using it in treatment of the management of a persistent epithelial defect as it is due to persistent inflammation and limbal stem-cell deficiency[11].

Autologous Serum:

In clinical trials topical fibronectin and epidermal growth factor helped in promoting reepithelialization. Autologous serum is prepared in clinical laboratories and are not commercially available.

SUPPORT REPAIR AND MINIMIZE ULCERATION

Ascorbic acid:

Vitamin C is a water-soluble vitamin and it is a cofactor in the rate-limiting step in collagen synthesis. There is decrease in secretion of ascorbate and a reduction in its concentration in the anterior chamber following damage to the ciliary body epithelium[12]. Vitamin C deficiency leads to impaired collagen synthesis in keratocytes. Although both systemic and topical ascorbate application have shown to reduce the occurrence of sterile corneal ulceration following chemical injury, topical application is superior. It has no effect after the stromal ulceration is established.

Collagenase Inhibitors:

Collagenase inhibitors like cysteine, acetylcysteine, sodium, calcium EDTA, and penicillamine prevents corneal stromal melting in severe chemical injuries. The disadvantage of collagenase inhibitors are poor corneal penetration, weak potency and instability[13].

Tetracycline derivatives have collagenase activity due to chelation of zinc at the active site of the collagenase enzyme. The most potent tetracycline collagenase inhibitory activity is Doxycycline[14].

CONTROL OF INFLAMMATION

Corticosteroids:

In acute and chronic inflammation the mainstay of treatment to reduce the tissue injury is Corticosteroids. Fear of the fact that re-epithelialization may be delayed and sterile corneal ulceration may occur following use of corticosteroids has resulted in a reluctance to use it in the acute phase.

Corticosteroids do not affect the rate of epithelial wound healing and it also decreases the inflammatory cell infiltration, which facilitates indirectly corneal epithelial migration and suppress sterile ulceration[15]. Corticosteroids do impair keratocyte migration and collagen synthesis and interfere with stromal repair[16] .

Corticosteroid use is successful in the first 7-10 days following chemical injury. Monitoring of corneal thinning should be done and steroids should be tapered. Later progestational steroids or nonsteroidal anti-inflammatory drugs (NSAID) can be used when corticosteroid related complications are more likely.

Progestational Steroids:

Medroxyprogesterone 1% has less anti-inflammatory potency than corticosteroids but has less deleterious effect on stromal repair and collagen synthesis. Medroxyprogesterone inhibits collagenase and reduces sterile ulceration after chemical injury[17]. It also suppresses corneal neovascularization. It is started after 10-14 days as a substituition to corticosteroids, as suppression of inflammation still is required, without the deleterious effect on stromal repair.

NSAIDs:

NSAIDs are an additive for corticosteroids in the first week and a substitute or additive for steroids later. The effect of NSAIDs wound repair, collagen synthesis, collagenolysis, and inhibition of neovascularisation is not yet clear.

SURGICAL THERAPY

Surgical intervention in the early phase is with Tenon's advancement, amniotic membrane transplantation, with or without limbal stem cell transplantation to achieve successful corneal re-epithelialization. Late surgical interventions available are penetrating or lamellar keratoplasty, for visual rehabilitation and structural integrity.

The identification of the role of stem cells in the regeneration and maintenance of the ocular surface is the most important advancement in the treatment of chemical injury[18]. The application of this knowledge in ocular surface transplantation techniques helps to specifically deal with stem cell loss and structural abnormalities at each phase of severe chemical injuries.

Ocular surface transplantation techniques like conjunctival and Tenon's advancement (tenoplasty), promote epithelial wound healing, helps in

reestablishment of limbal vascularity and provides source of epithelium for the denuded corneal surface[19]. Amniotic membrane grafting increases the migration of surviving limbal stem cells. For reestablishment of limbal stem cells, limbal stem-cell transplantation can be done. Mucosal membrane transplantation is used to form the conjunctival fornices and normal apposition of lid and globe. For the success of corneal transplantation for visual rehabilitation, reestablishment of normal corneal epithelium and normal conjunctival fornix and lid-globe relationships is essential.

TISSUE ADHESIVE :

Tissue adhesive are useful in preventing further progression of sterile corneal ulceration. It is also used in maintaining the integrity of the globe, when other measures have failed. It is best for use for impending or actual perforations, that are 1mm or smaller in size and it is preferred to emergency tectonic procedures. A tectonic penetrating keratoplasty may be used to preserve the integrity of the globe for perforations that are (>1 mm) that cannot be adequately addressed with tissue adhesive. After 6-8 weeks the tissue adhesive may be removed or allowed to extrude spontaneously, when a stable fibrovascular scar has formed and when the risk of subsequent stromal ulceration is eliminated. This fibrovascular scar may impair vision

and worsen the prognosis for subsequent penetrating keratoplasty unfortunately.

AMNIOTIC MEMBRANE TRANPLANTATION:

Human amniotic membrane is the thin, innermost layer of the placenta. It consists of an epithelial monolayer, an avascular stromal matrix and a thick basement membrane. The tissue may be transplanted with the basement membrane upward or downwards to the corneal surface. The amniotic membrane behaves like a biological bandage contact lens when used with the basement membrane oriented downwards thereby promoting epithelialization under the membrane[20]. It dissolves earlier than desired before reepithelialization is complete. When it is used with basement membrane oriented upward, then it behaves like an inlay graft, with the amniotic membrane functioning as a new basement membrane which promotes epithelialization over its surface[21]. It becomes incorporated into the substratum of the newly formed epithelium and persists for months. But it does not always possess the same transparency of healthy stroma. It can also be used with a combination of an inner amniotic membrane with the basement membrane oriented upward and an outer amniotic membrane with the basement membrane oriented downward and the reepithelialization is 'sandwiched' between two basement membranes.

Irrespective of the transplantation technique, reepithelialization is facilitated by amniotic tissue if complete or partial limbal stem-cell function is present. Reepithelialization is improved by providing a basement membrane for initiation of epithelial migration and adhesion and it also releases growth factors that facilitate proliferation of limbal stem cells and transient amplifying cells. Amniotic tissue by excluding inflammatory cells from the corneal stroma can reduce inflammation and it also releases factors that suppress proinflammatory cytokines, facilitate apoptosis of inflammatory cells, and reduce epithelial and keratocytes apoptosis. It also reduces corneal neovascularisation and conjunctival scar formation after chemical injury.

The use of amniotic membrane in the treatment of ocular surface disease was reintroduced by Kim and Tseng in 1995. Treatment of acute phase grade II and III injuries with both onlay and inlay graft techniques results in successful reepithelialization.

In the treatment of grade IV injuries, disappointing results have been reported, due to total limbal stem-cell loss.

Amniotic membrane transplantation may be used in the late rehabilitation phase, either alone or in conjunction with limbal stem-cell transplantation. It is effective in cases of persistent epithelial defects, recurrent epithelial erosions, and in the reduction of chronic inflammation. It is also combined with reconstructive procedures of the ocular surface like symblepharon lysis. In cases of complete limbal stem-cell function, it is used as adjunctive with limbal stem-cell transplantation.

LIMBAL STEM-CELL TRANSPLANTAION

Kenyon and Tseng developed limbal stem-cell transplantation as a modification of Thoft's original conjunctival transplantation technique[22]. In the clinical course of a grade III or IV injury it is the best method of reestablishing a phenotypically correct corneal epithelial surface and preventing the problems of fibrovascular pannus or sterile corneal ulceration. In addition, this procedure may be performed later in the clinical course in order to improve ocular surface function. It can also be a part of a staged procedure with penetrating keratoplasty to provide visual rehabilitation.

In unilateral or asymmetric cases of chemical injury, conjunctival limbal autograft transplantation (CLAU) is usually performed by harvesting contralateral limbal stem cells from the unaffected or less injured fellow eye and transferring them to the affected or more injured eye. Ipsilateral CLAU is an option for treatment of limited sectorial limbal stem-cell deficiency, where the transfer of tissue from an unaffected area in the opposite meridian of the damaged limbus to the ischemic area is done[23].

Limbal allograft transplantation from a relative or a cadaver donor are the only viable options in cases of severe bilateral injures. The procedure of Living-related conjunctival limbal allograft transplantation (Ir-CLAG) is technically same as CLAU with the exception that the limbal stem cells are harvested from a close relative and transferred to the injured eye[24]. The risk of graft rejection is higher after this procedure than with keratoplasty mostly due to the transplantation of vascular tissue into a vascular bed and a higher concentration of transplanted antigens in the peripheral cornea.

The use of prolonged topical and systemic immunosuppression prevents rejection and maintenance of a stable stem cell population. Use of topical corticosteroids and cyclosporine indefinitely or triple

immunosuppressive therapy with tapering doses of prednisone, cyclosporine A, and azathioprine for a minimum of 12-24 months are recommended. Rejection rates as high as 25-33% have been reported despite systemic immunosuppression. Keratolimbal allograft transplantation (KLAT)is used for transferring the limbal stem cells from a donor cadaver for treatment of severe bilateral chemical injuries. The risk of graft rejection is very high, but with prolong, aggressive topical and systemic immunosuppression successful preservation of limbal stem-cell function and corneal clarity has been reported

Expansion of limbal stem cells ex vivo is being investigated as an improvement of existing limbal stem-cell transplantation techniques. In this procedure, the small piece of donor limbal tissue is dissected and placed in a cell culture. After the growth and expansion of viable limbal stem cells in culture, transplantation of the epithelial sheet to the recipient eye is performed. The technical difficulties in cell culture and transfer of the epithelial sheet to the recipient eye are the limitations in the application of this technique. In future it will be possible to restore the function in all cases of severe bilateral injuries by autograft transplantation, without the risk of immunological rejection. If antigen-presenting Langerhan's cells are

eliminated during cell culture and if only epithelial cells are transplanted in cases where harvesting of limbal stem cells is required from a living relative or a cadaver, the risk of rejection will be reduced.

MUCOSAL MEMBRANE TRANSPLANTATION

Progressive scarring of the bulbar and palpebral conjunctiva leads to mechanical abnormalities like restriction of extraocular movement, fornix foreshortening and obliteration, symblepharon formation and lid abnormalities like incomplete lid closure, cicatricial entropion, trichiasis, and lid margin keratinization. Bulbar conjunctival transplantation in unilateral cases can correct most of these abnormalities. In bilateral cases use of mucosal membrane grafts to reconstruct the fornix and restore normal lid-globe relations, prevents the above mentioned complications. But they do not restore the corneal epithelial functions following limbal stem-cell transplantation. The impaired goblet cell function of the conjunctiva can be improved by harvesting mucosal grafts from nasal mucosa.

PENETRATING KERATOPLASTY

After the rehabilitation of the ocular surface has been achieved, an optical penetrating keratoplasty may be attempted[25]. Performing limbal stem-cell transplantation prior to penetrating keratoplasty or doing the procedures simultaneously may facilitate more rapid visual rehabilitation. The prognosis for successful penetrating keratoplasty depends on the original severity and sequelae of the chemical injury and the adequacy of restoration of normal ocular surface.

Kuckelkorn and Redbrake have reported success with large-diameter(11 to 12mm) penetrating keratoplasty in both the acute and chronic chemical injuries[26]. In the acute setting, penetrating keratoplasty is highly successful in the management of impending or actual corneal perforation, but in the long-term prognosis for graft clarity is poor. When the procedure is done for combined late rehabilitation of persistent limbal stem-cell dysfunction and corneal opacification, the prognosis for graft survival is poor, mainly because of the allograft rejection of the donor limbal stem-cells, rather than endothelial rejection.

KERATOPROSTHESIS

Kerotoprosthesis may be useful in bilateral, several chemical injury with irreparable damage to the ocular surface in which the prognosis is hopeless for penetrating keratoplasty. It can also be tried in unilateral cases with repeated immunologic endothelial rejection. The success rate of keratoprosthesis has been poor in the past. The success rate has improved following improved Keratoprosthesis design and better postoperative management available now.

TENOPLASTY

TENOPLASTY / POSTERIOR TENON'S ADVANCEMENT

Due to severe limbal ischemia in eyes with chemical injury, persistent conjunctival or corneal defect develop and there is high risk of perforation. The surgical approach of bringing back the blood supply to the ischemic area by placing the vital connective vascular tissue of the orbit, is described in 1989 by Teping C and Reim M and is known as tenon's advancement or tenoplasty[27]. The following surgical technique uses an amniotic membrane as a substrate for the relocation of vital vascular tenon sheet.

Surgical Technique :

A peritomy is done to separate the necrotic tissue from the remaining healthy conjunctiva with the help of corneal scissors. The necrotic tissue is dissected and removed with corneal scissors. After the removal of necrotic tissue, a free pedicle tenon vascular sheet is dissected from the deep posterior part of the unburned area. It is done by creating a plane by between the conjunctiva and the subconjunctival fibrovascular tissue using 0.12 forceps, and this plane can be further dissected to separate the conjunctiva from the subconjunctiva, by using scissors. After the separation of the conjunctival layer from the subconjunctival tissue, a second plane is identified between the deep tenon layer and subconjunctival

fibrovascular tissue by scissors. A free vascular pedicle graft can be moved from the posterior pole towards the limbus. Once an adequate size of pedicle tenon vascular sheet obtained, the bare sclera is protected by using a layer of amniotic membrane with the stromal side face down and it is anchored to the sclera by using interrupted 8-0 Vicryl sutures at the conjunctiva and 10-0 nylon at the limbus along with episcleral bites. The free pedicle vascular tenon sheet is placed over the amniotic membrane and secured to it with interrupted 8-0 Vicryl sutures with episcleral bites and a second layer of amniotic membrane is used to cover the vascular tenon sheet and secured with 10-0 nylon at the limbus and with 8-0 Vicryl sutures at the conjunctiva. The amniotic membrane is secured to the conjunctiva and to the lid margin with the stromal or sticky side facing down by using 8-0 or 9-0 Vicryl sutures with episcleral bites. At the lower and upper fornices the amniotic membrane is further secured with two double-arm 4-0 silk sutures, which are brought through the lid and tied over the skin with bolsters. At the end, the entire ischemic area is protected by a sandwiched vascular tenon sheet with two amniotic membrane grafts.

PART II

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

The aim of the study is to study the following parameters

1. The effect of tenoplasty in healing of severe chemical injury
2. The effect of tenoplasty in improvement of limbal ischemia
3. The effect of tenoplasty in preserving the ocular surface integrity
4. The clinical presentation and course of severe chemical injury

PATIENT SELECTION

INCLUSION CRITERIA:

All patients with severe chemical injuries (acid and alkali injuries) – grade IV and above by DUA Classification

Duration of the study - 2 years

EXCLUSION CRITERIA:

1. Patients with corneal perforation
2. Patient mild chemical injury (grade III and less by DUA classification)
3. Patients with infected corneal ulcers following chemical injury

DUA's CLASSIFICATION OF CHEMICAL INJURY

Grade	Clinical findings	Conjunctival involvement	Prognosis
I	0 clock hours of limbal involvement	0%	Very good
II	<3 clock hours of limbal involvement	<30%	Good
III	>3–6 clock hours of limbal involvement	>30–50%	Good
IV	>6–9 clock hours of limbal involvement	>50–75%	Good to guarded
V	>9–<12 clock hours of limbal involvement	>75–<100%	Guarded to poor
VI	Total limbus (12 clock hours) involved	Total conjunctiva (100%) involved	Very poor

PRE-OPREATIVE SCREENING PROCEDURES

Patients are screened for Visual acuity by Snellen's chart , intraocular pressure evaluation by schiotz tonometer or noncontact tonometer, Slit lamp examination, Fundus examination, Blood pressure, glycemic indices like serum glucose levels and urine- alb/sugar.

POST-OPERATIVE VISITS

Patients are reviewed on the 1st post operative day, then on everyday till the re is epithelial defect healing and the occurrence of limbal vasculature. The findings are documented at 1 week and at 1 month.

MATERIALS AND METHODS

MATERIALS AND METHODS

Patients attending casuality with chemical injury are first given copious amount of saline wash with normal saline for thirty minutes. The residual chemical debris stuck on to cornea and conjunctiva are removed with the help of cotton bud or surgical blade. The patients are subjected to brief history regarding the time of injury, type of chemical, mode of injury. Visual acuity of the patient is documented. The patients are examined in slit lamp for the extent of chemical injury and graded based on the amount of limbal ischemia in clock hours and amount of conjunctiva exposed in percentage of tissue and graded according to dua's classification.

Medical management with topical steroids like 1%prednisolone acetate, lubricant 1% carboxy methylcellose, preservative free antibiotic eye drops like moxifloxacin, vitamin c eye drops. Patients are also started on oral vitamin c tablets and oral doxycycline for collagenolytic activity. Patients with severe chemical injury that is grade 4 or more by dua's classification are taken up for tenoplasty.

SURGICAL TECHNIQUE :

Under local anaesthesia, peritomy is done to separate the necrotic tissue from the remaining healthy conjunctiva with the help of corneal scissors. The necrotic tissue is dissected and removed with corneal scissors. After the removal of necrotic tissue, a free pedicle tenon vascular sheet is dissected from the deep posterior part of the unburned area. It is done by creating a plane by between the conjunctiva and the subconjunctival fibrovascular tissue using 0.12 forceps, and this plane can be further dissected to separate the conjunctiva from the subconjunctiva, by using scissors. After the separation of the conjunctival layer from the subconjunctival tissue, a second plane is identified between the deep tenon layer and subconjunctival fibrovascular tissue by scissors. A free vascular pedicle graft can be moved from the posterior pole towards the limbus. Once an adequate size of pedicle tenon vascular sheet obtained, the bare sclera is protected by using a layer of amniotic membrane with the stromal side face down and it is anchored to the sclera by using interrupted 8-0 Vicryl sutures at the conjunctiva and 10-0 nylon at the limbus along with episcleral bites. The free pedicle vascular tenon sheet is placed over the amniotic membrane and secured to it with interrupted 8-0 Vicryl sutures with episcleral bites and a second layer of amniotic membrane is used to cover the vascular tenon sheet and secured with 10-0 nylon at the limbus

and with 8-0 Vicryl sutures at the conjunctiva. The amniotic membrane is secured to the conjunctiva and to the lid margin with the stromal or sticky side facing down by using 8-0 or 9-0 Vicryl sutures with episcleral bites. At the lower and upper fornices the amniotic membrane is further secured with two double-arm 4-0 silk sutures, which are brought through the lid and tied over the skin with bolsters. At the end, the entire ischemic area is protected by a sandwiched vascular tenon sheet with two amniotic membrane grafts.

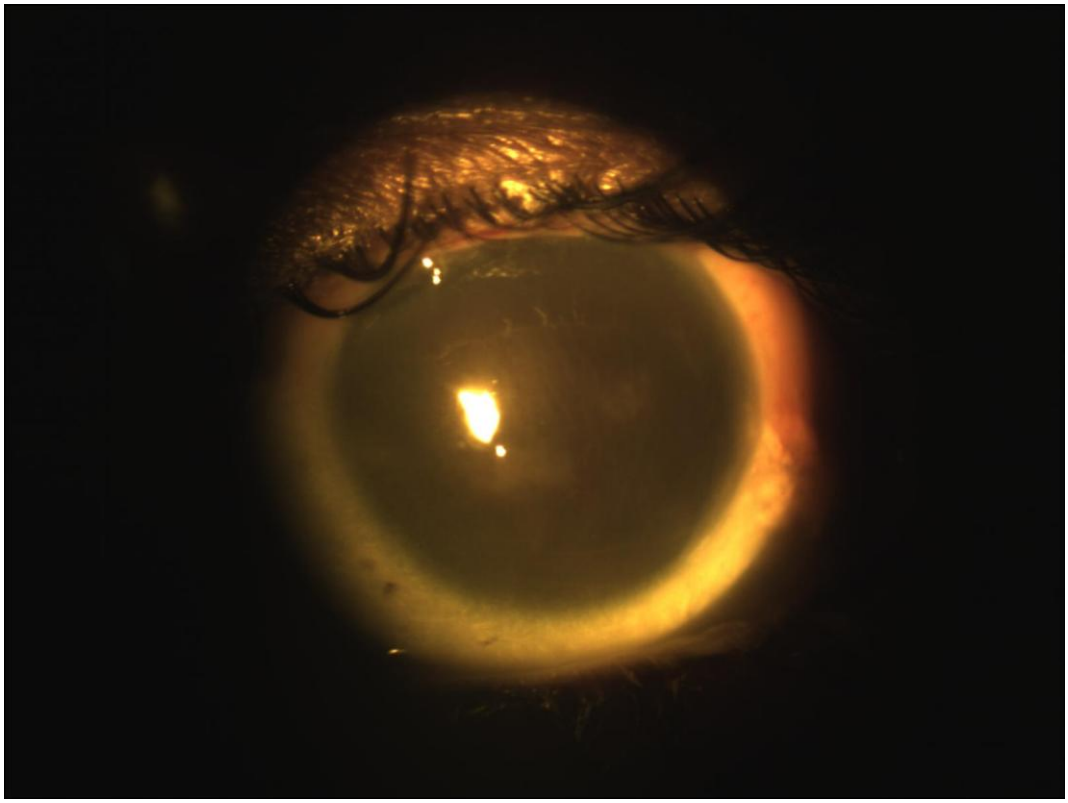


Fig 1 LIMBAL ISCHEMIA

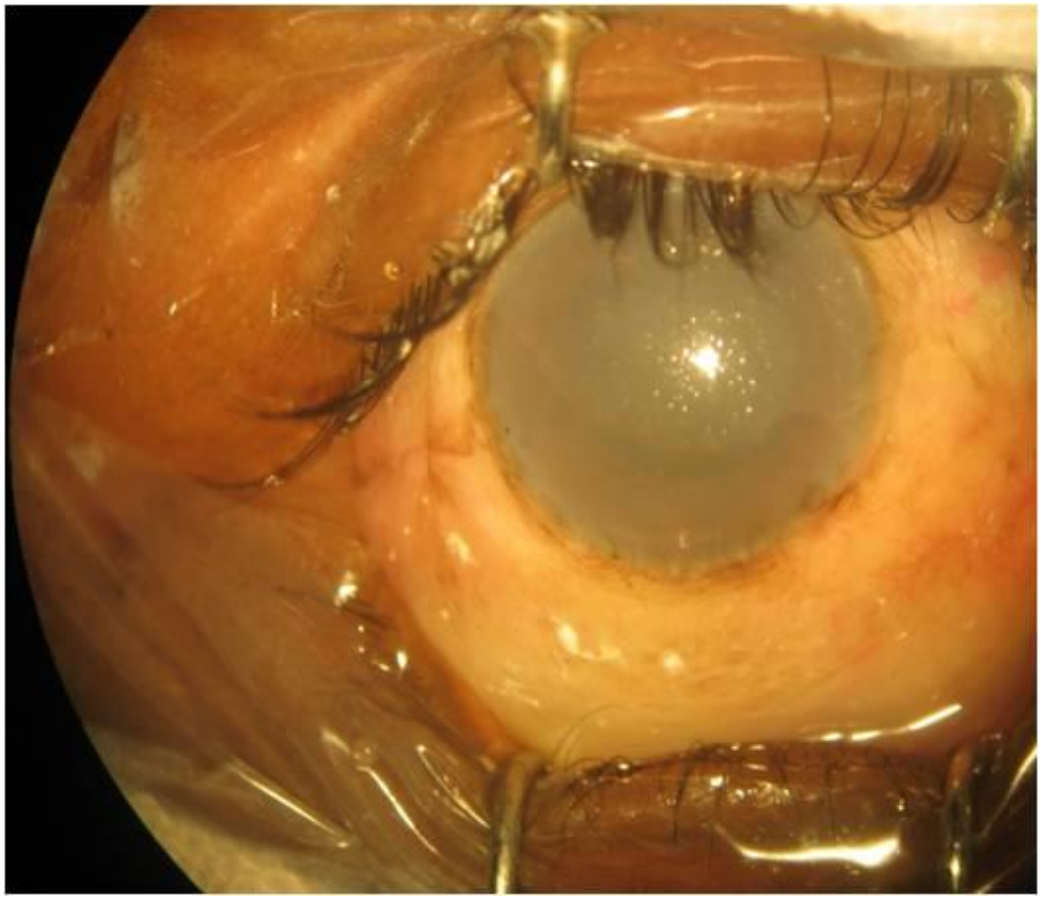


Fig 2 CONJUNCTIVAL CHEMOSIS

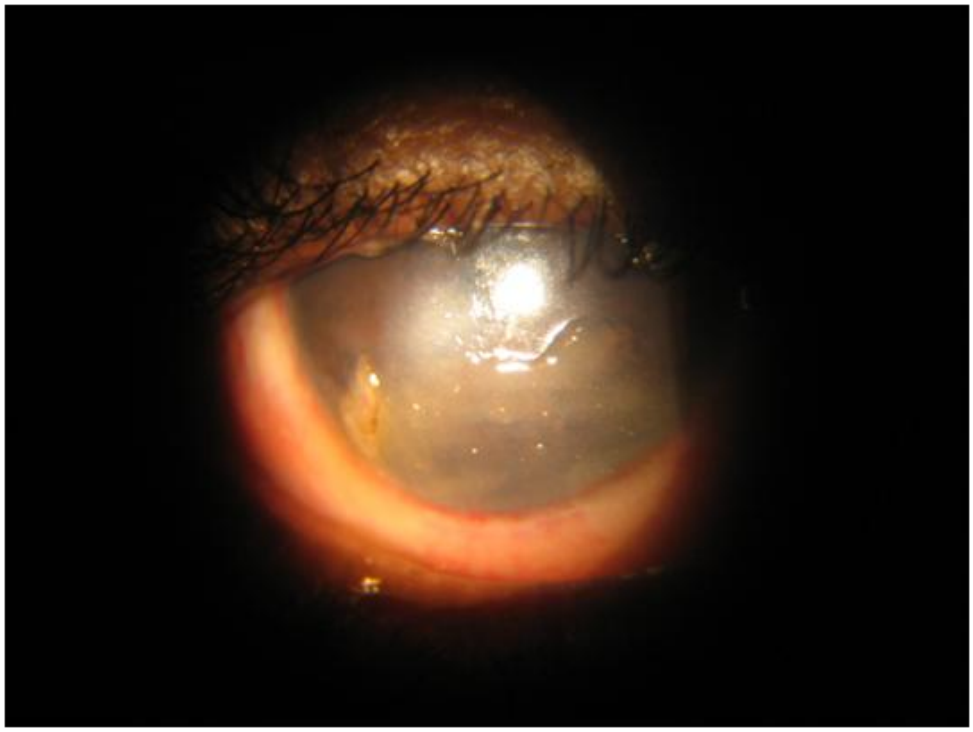


Fig 3 EPITHELIAL DEFECT

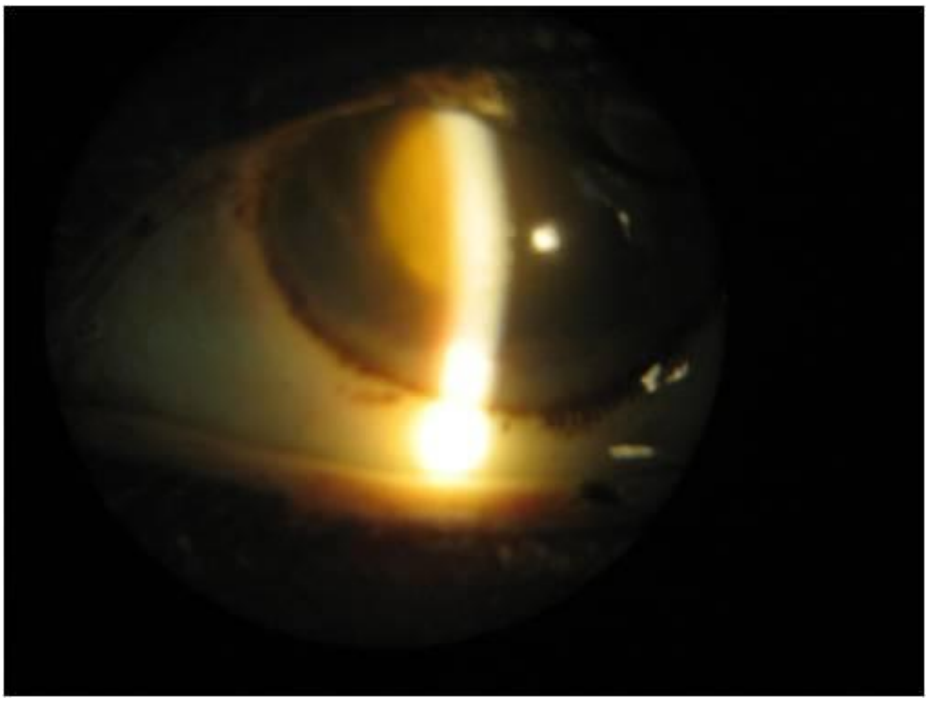


Fig 4 GRADE VI CHEMICAL INJURY

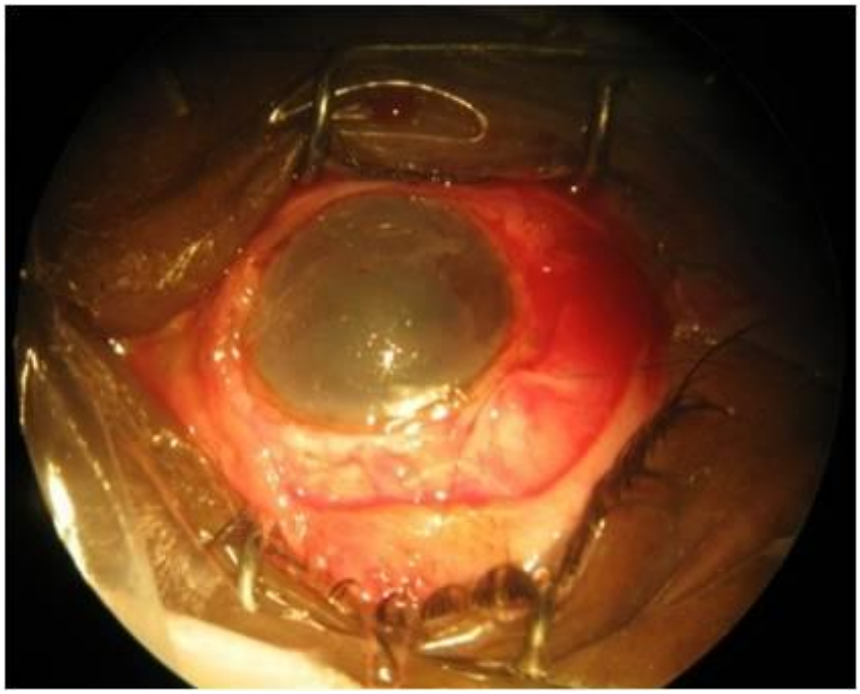


FIG 5 TENOPLASTY

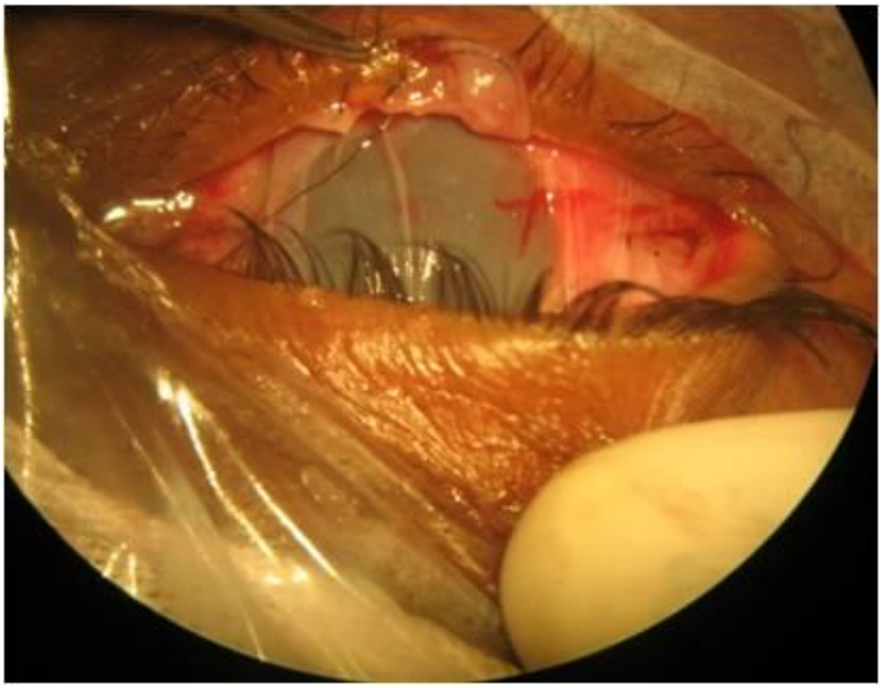


FIG 6 AMG OVER TENOPLASTY

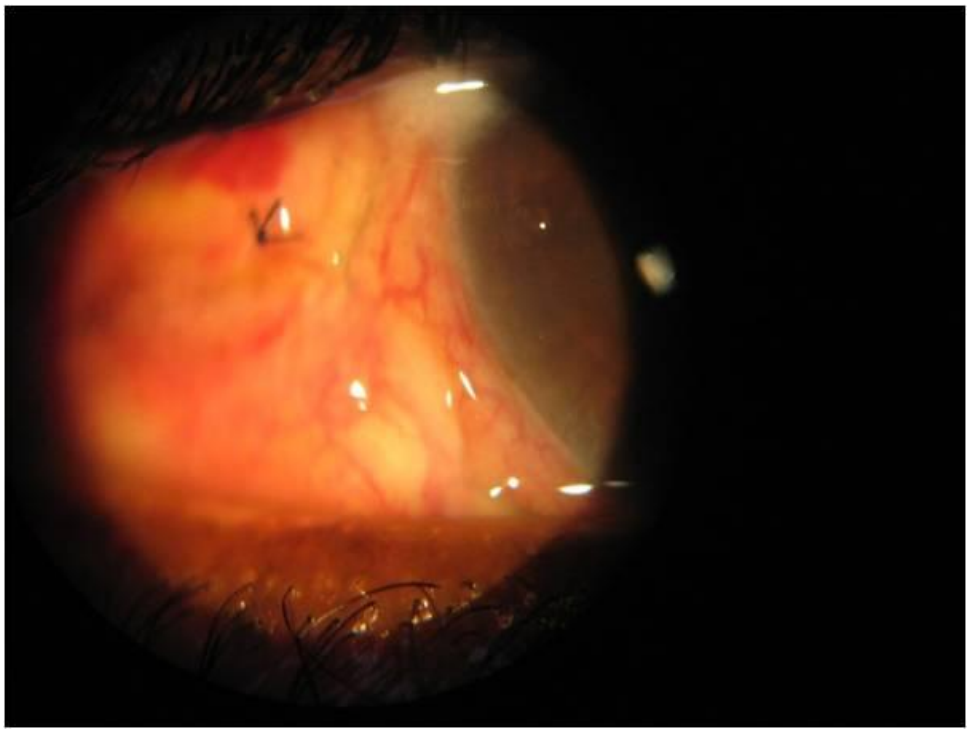


FIG 7 IMMEDITE POSTOPERATIVE PICTURE

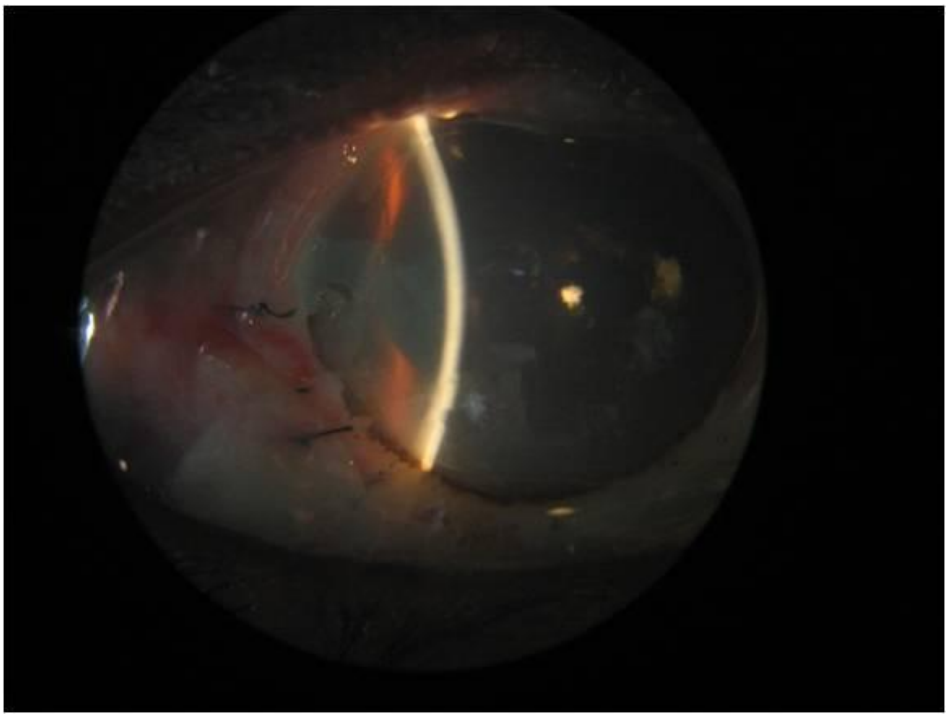


FIG 8 SUTURES AT THE LIMBUS

FIG 9 SYMBLEPHARON FOLLOWING CHEMICAL INJURY

RESULTS

RESULTS

Total number of eyes in the study- 20 eyes of 14 patients

Mean age of the patients – 27.07 years

TABLE [1]

SEX RATIO

Sex	Number of patients	Percentage
Male	7	50%
Female	7	50%

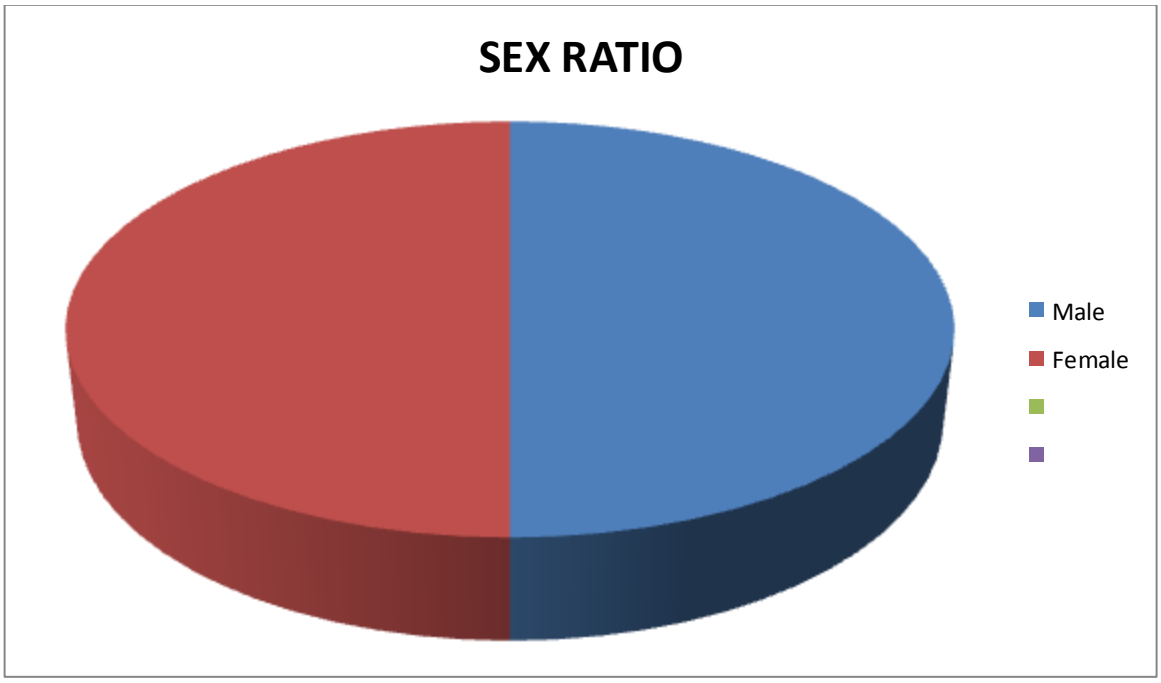


TABLE [2]

LATERALITY

Laterlity	Number of patients	Percentage
Both eyes	6	42.86
Right eye	3	21.43
Left eye	5	35.71

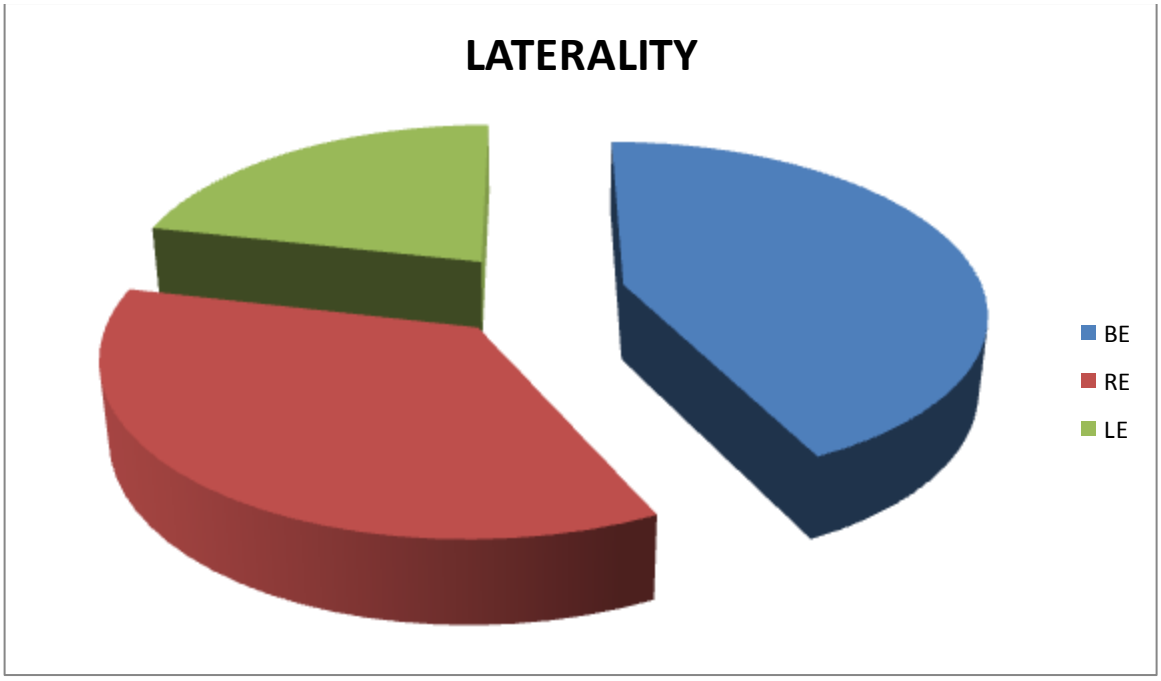


TABLE [3]

MODE OF INJURY

Mode of injury	Number of patients	Percentage
Accidental	8	57.14
Assault	4	28.57
Industrial	2	14.28

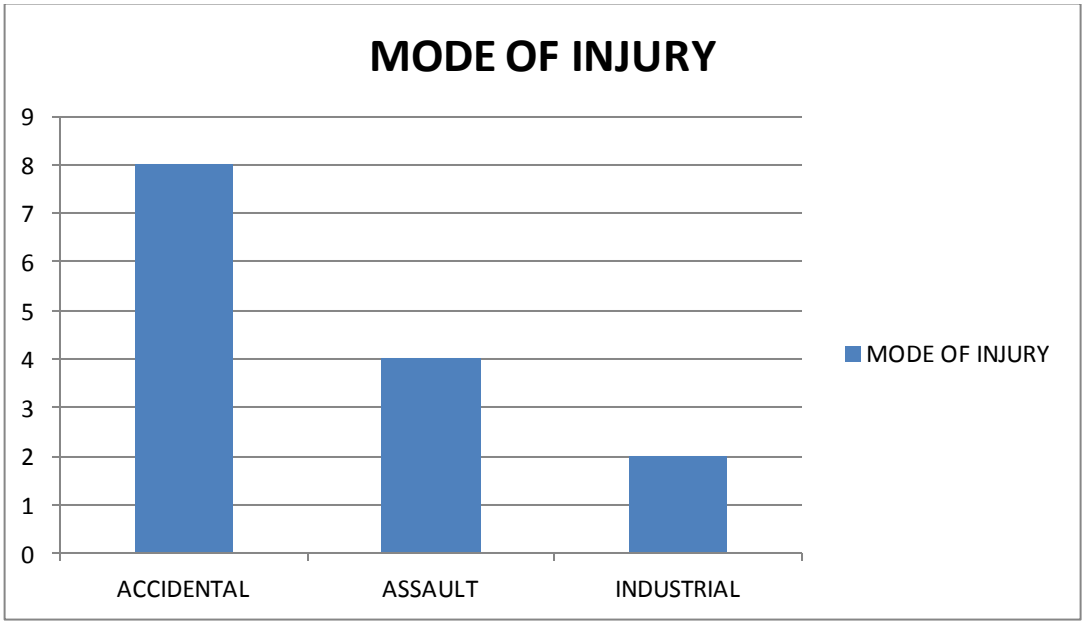


TABLE [4] TYPE OF CHEMICAL

Chemical	Number of patients	Percentage
Acid	8	57.14
Alkali	6	42.86

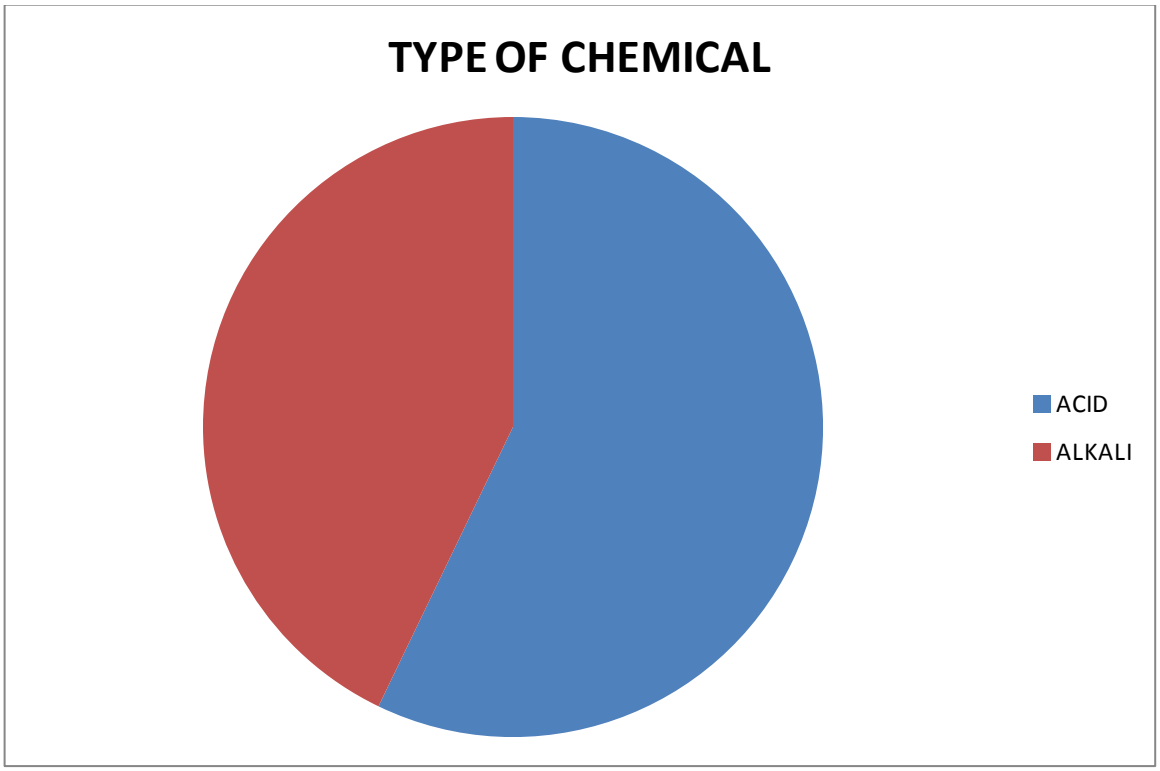


TABLE [6]

MEAN VISUAL ACUITY

Range of visual acuity at presentation	Number of eyes	Percentage
>5/60	8	40%
1/60 – 5/60	5	25%
<1/60	6	30%

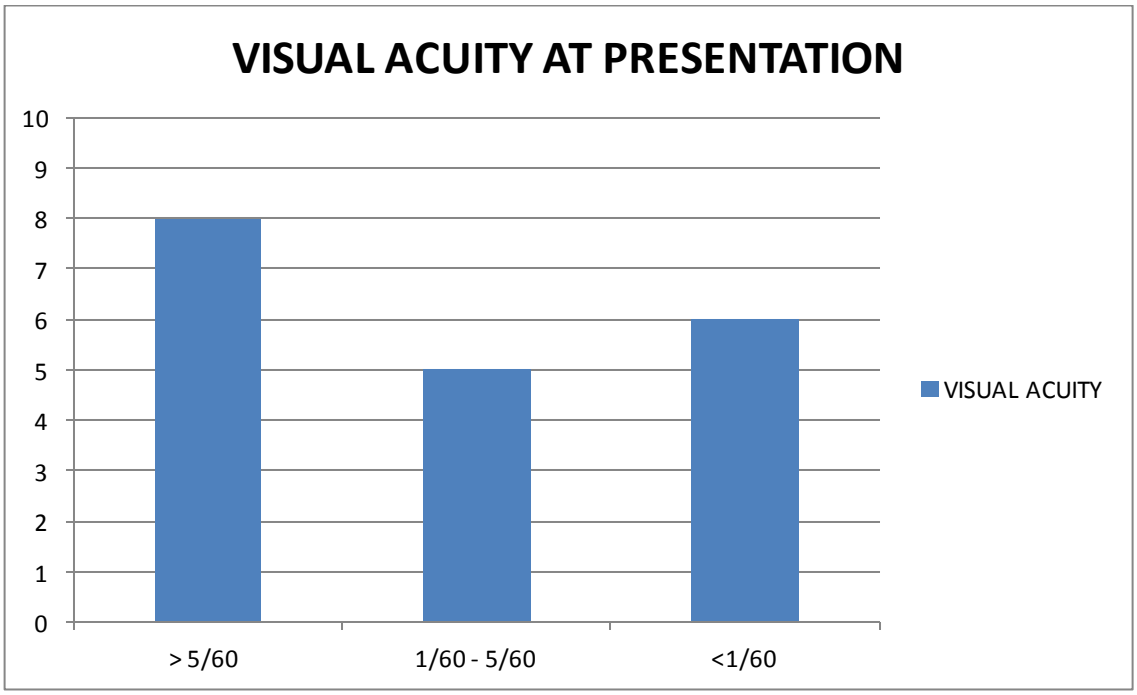


TABLE [7]

MEAN LIMBAL ISCHEMIA

Limbal ischemia in clock hours	Number of eyes	Percentage
6	9	45%
7	3	15%
8	3	15%
9	2	10%
10	2	10%
12	1	5%

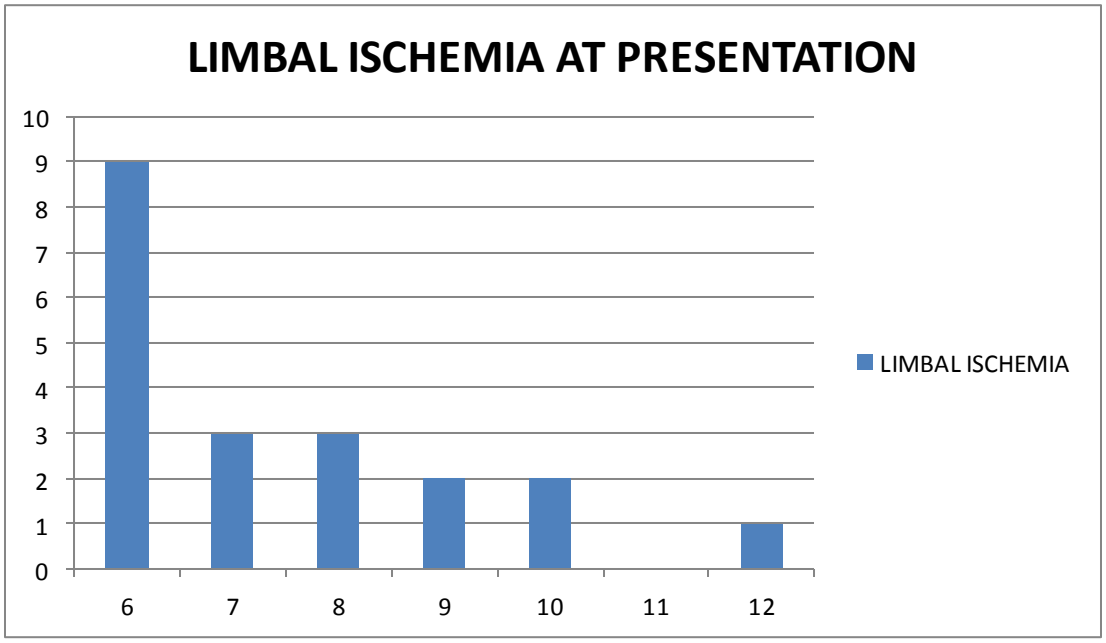
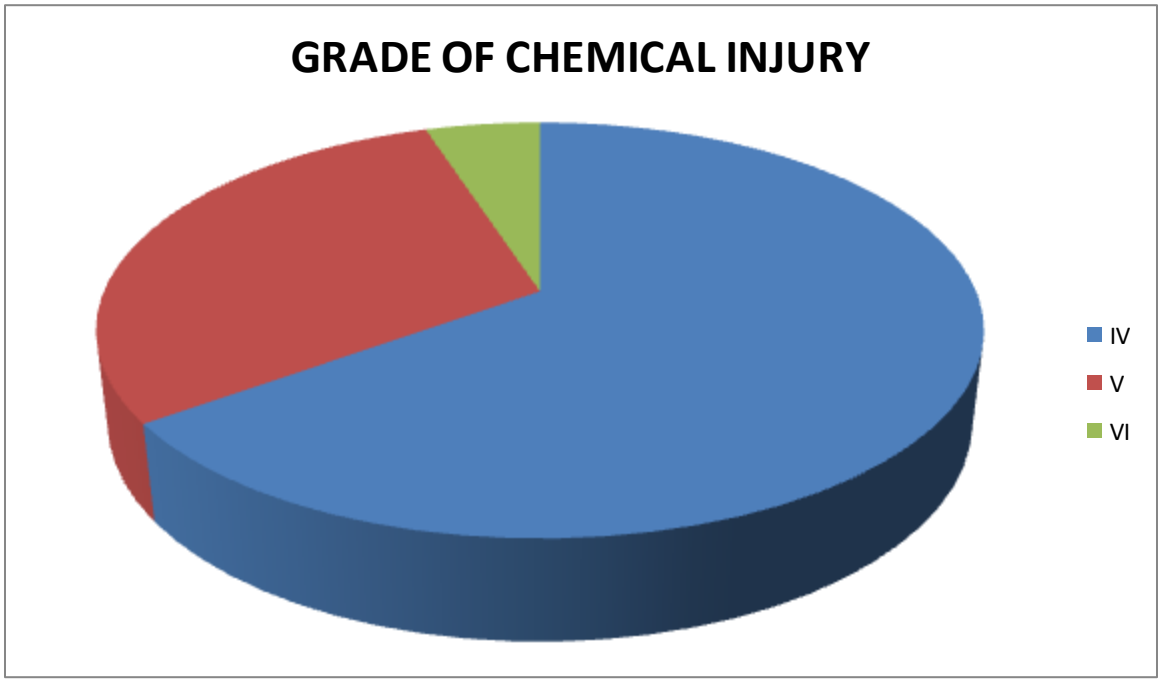


TABLE [8]

GRADE OF CHEMICAL INJURY

Grade of injury	Number of eyes	Percentage
IV	13	65%
V	6	30%
VI	1	5%



Mean time of surgery – 4.6 days

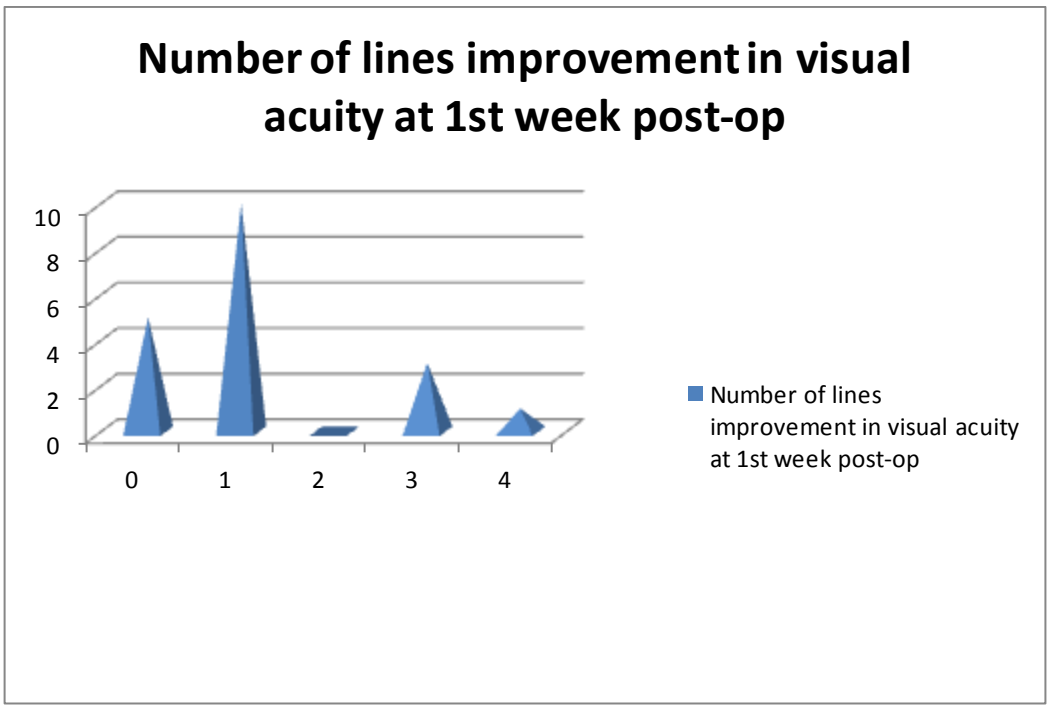
Epithelial defect was present in all cases

TABLE [9] IMPROVEMENT IN VISUAL ACUITY 1 WEEK FOLLOWING TENOPLASTY

Number of lines in snellens chart	Number of eyes	Percentage
Nil	5	25
=<2	10	50
>3	4	20

TABLE [10] IMPROVEMENT IN VISAUL ACUITY IN VARIOUS GRADES OF CHEMICAL INJURY

Number of lines improvement in visual acuity	Grade IV	Grade V	Grade VI
0	1	2	0
1-2	8	2	0
3-6	4	1	1


Number of lines improvement in visual acuity at 1st week post-op
10
8
6
4
2
0
0
1
2
3
4
Number of lines improvement in visual acuity at 1st week post-op

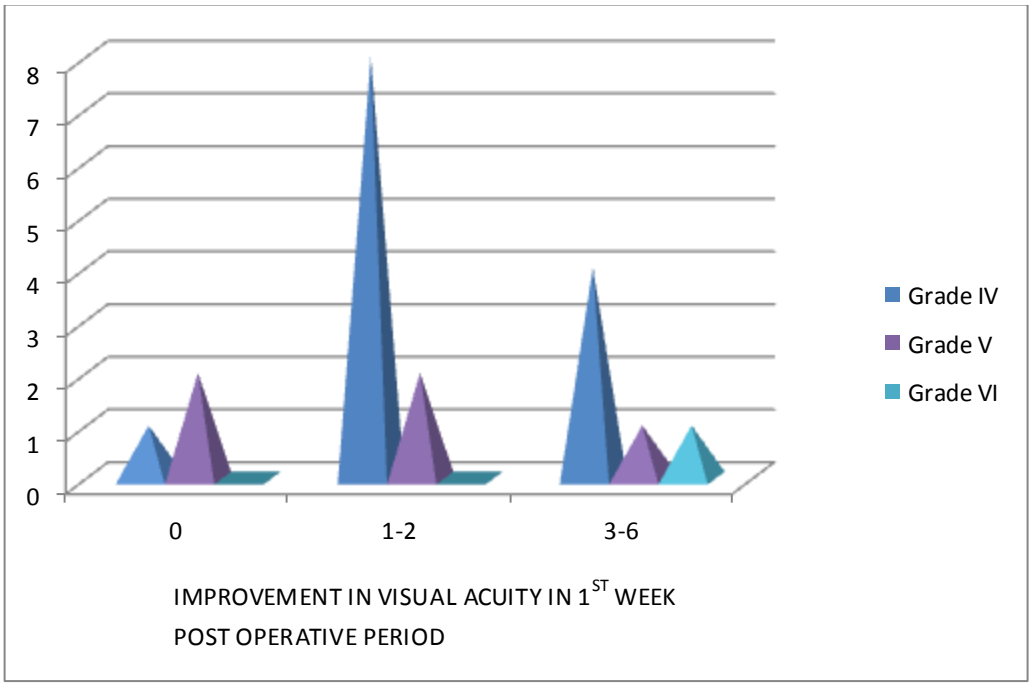

8
7
6
5
4
3
2
1
0
0
1-2
3-6
Grade IV
Grade V
Grade VI
IMPROVEMENT IN VISUAL ACUITY IN 1ST WEEK POST OPERATIVE PERIOD

TABLE [11] IMPROVEMENT OF LIMBAL ISCHEMIA AT 1ST WEEK POST-OP

Improvement in limbal ischemia in clock hours	Number of eyes	Percentage
4	3	15
5	2	10
6	11	55
7	2	10
8	2	10

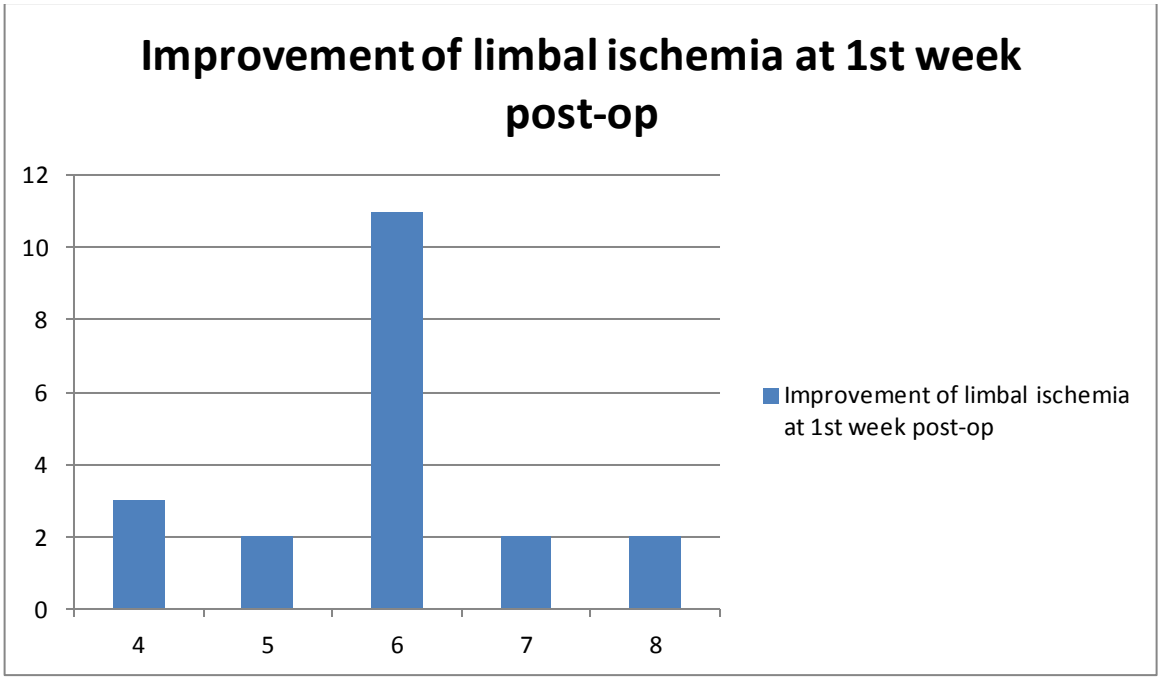


TABLE [12]

IMPROVEMENT IN VISUAL ACUITY AT 1 MONTH POST-OP

Number of lines improvement in visual acuity	Number of eyes	Percentage
0	3	15%
=<2	6	30%
>3	10	50%

IMPROVEMENT IN LIMBAL ISCHEMIA AT 1 MONTH POST-OP

At the end of one month all patients of grade IV and grade V had no residual limbal ischemia. One patient belonging to grade VI chemical injury developed residual limbal ischemia at the end of one month

SYMBLEPHARON FORMATION

Symblepharon formation was found in one patient(5%) of the study

DISCUSSION

DISCUSSION

Our study was conducted in a tertiary eye care in south india, from august 2010 to September 2012, included 20 eyes of 14 patients. The mean age of presentation of our study group was 27.07 years which included two pediatric patients also. It was similar to the *Adepoju et al* where the mean age of the study group was 31.87 years, emphasising the prevalence of chemical in the young working population[28].

In our study the male-female preponderance was equal in both the groups. But most of other studies like *Adepoju et al*[28] and *Le et al*[29], showed more male preponderance as they included all grades of chemical injuries whereas in our study only the severe chemical injury patients were enrolled.

In our study, 57% of patients the mode of chemical injury was accidental. 28% of patients developed chemical injury by assault and 14% of injuries were industrial accidents. In other studies like Xie et al, majority of the chemical injuries were occupational[30]. Even in our study most of the accidental injuries were due to household cleaning acid.

In our study 42.1% of patients had bilateral chemical injuries. Among the unilateral cases the prevalence of left eye injuries were more than right eye injuries. Other studies like Saini et al[31] also show similar bilateral involvement with 42.9%. In our study, acid injuries (57%) were slightly more common than alkali injuries(43%). Other studies like Tejwani et al, showed more prevalence of alkali injuries[32].

In our study which dealt only with severe chemical injuries, 40% of the eyes had visual acuity of =or>6/60. 20% of the eyes had visual acuity of the range 1/60 -5/60 by Snellens chart. 35% of the patients had visual acuity <1/60. The maximum visual acuity of the patient enrolled was in two patients with 6/18, both of them had 6 clock hours of limbal ischemia. The minmum visual acuity of the patient in the study was PL in two patients with total limbal ischemia with cataract and they did not improve following tenoplasty. In the study by *Adepoju et al*[28] ,33.3% of patients had vision <3/60 which is similar to our study.

In our study patients were classified based on Dua's classification which classifies patient based on limbal ischemia and area of conjunctival involvement. Limbal ischemia and conjunctival chemosis represent the amount of exposure to the chemical and hence they have prognostic value.

Gupta et al showed that, Dua classification by providing further subclassification of grade IV chemical injuries by Roper Hall into three separate grades has a superior prognostic predictive value in severe chemical injuries[33].

In our study 13 eyes(65%) belonged to grade IV, 6 eyes had grade V clinical features and one eye had grade VI clinical features of limbal ischemia and conjunctival involvement. Tenoplasty was done in all patients. Two eyes of two pediatric patients were taken for surgery under general anaesthesia and the rest of the patients were operated in peribulbar block.

The mean time of delay in surgical treatment was 4.6 days. Earliest surgery was done on the same day of the injury in a grade IV acid injury patient, who on follow up had complete regression of limbal ischemia. Maximum delay in surgery for the primary eye was 7 days, in both the pediatric patients due to the delay in obtaining fitness for general anaesthesia, and in one patient with grade VI acid injury, the delay was due to uncontrolled diabetes.

At the end of one week post-op, among the eyes with grade IV chemical injury(13), in 1 patient(7.6%) there was no improvement in visual acuity, 8 eyes(61.5%) had <3 line improvement in snellens chart and 4 eyes had >2 line improvement in visual acuity, the maximum being 5 line improvement in one patient. In eyes with grade V injuries(5), 2(40%) eyes had no visual improvement , 2(40%) eyes had <3 line visual acuity improvement and 1(20%) patient had >2 line improvement in visual acuity. One of a patient had grade VI in the study and she didnot show any improvement in visual acuity in 1st week.

The improvement in limbal ischemia in the study at the end of one week post-operative period was as follows. 16(80%) eyes had improvement in limbal vascularisation of < or= 6 clock hours, 4(20%) eyes had improvement >6 clock hours. 9 (69.3%) out of 13 eyes with grade IV injury did not have residual ischemia at the end of one week. In 5 eyes with grade V injuries 2 eyes had residual limbal ischemia of 2 clock hours and in 3 patients, of more than 3 clock hours. In one eye with grade VI injury the residual limbal ischemia was 6 clock hours.

At the end of one month, in 13 eyes grade IV injuries, improvement in visual acuity = or>3 lines was seen in 8(61.5%) patients, 4 eyes had visual

acuity improvement of <3 lines, and one eye had no improvement in visual acuity due to development of cataract. These findings were similar to the results obtained at 1st week postoperative period.

All patients of grade IV and V chemical injury had no residual limbal ischemia or ocular surface abnormality at the end of one month. One patient who presented with grade VI injury had a residual ischemia of 4 clock hours with symblepharon formation. In a study done by Tamhane et al with only amniotic membrane transplantation there was no overall difference in the final visual acuity, symblepharon formation, corneal vascularization, and tear function tests between medical treatment and amniotic membrane grafting[34]. Sharma et al showed improvement in visual acuity and preservation of ocular surface integrity with the help of tenoplasty with cyanoacrylate glue[35]. In another study done by Kuckelkorn R et al on tenoplasty for severe chemical injuries, in all patients, corneoscleral ulceration was prevented and ocular surface integrity was maintained[36]. Thus amniotic membrane transplantation alone improves the corneal epithelial healing but has no improvement in limbal stem cell deficiency, whereas tenon's advancement improves the limbal vasculature and prevents corneoscleral ulceration and maintains the integrity of the globe for future optical keratoplasty.

SUMMARY

SUMMARY

A total of 20 eyes of 14 patients were included in our study with severe chemical injury based on Dua's classification. All patients underwent tenoplasty. The area of limbal ischemia, conjunctival involvement, visual acuity and other associated complications were noted.

- The mean age of presentation of our study group was 27.07 years

- The male-female preponderance was equal in both the groups in the study.

- The mode of injury in our study was, 57% of patients the mode of chemical injury was accidental. 28% of patients developed chemical injury by assault and 14% of injuries were industrial accidents.

- Most of the accidental injuries were due to household cleaning acid.

- 42.1% of patients had bilateral chemical injuries

- In the unilateral cases the prevalence of left eye injuries was more than right eye injuries

- Acid injuries (57%) were slightly more common than alkali injuries(43%).

- At the time of presentation, 40% of the eyes had visual acuity of =or>6/60. 20% of the eyes had visual acuity of the range 1/60 -5/60 by Snellens chart. 35% of the patients had visual acuity <1/60.

- By Dua's classification, 65% belonged to grade IV, 30% of eyes had grade V clinical features and 5% had grade VI.

- The mean time of delay in surgical treatment was 4.6 days

- At the end of one week post-op, among the eyes with grade IV chemical injury 61.5% had <3 line improvement in snellens chart and 30.75% eyes had >2 line improvement in visual acuity

- In eyes with grade V injuries 40% eyes had no visual improvement , 40% eyes had <3 line visual acuity improvement and 20% eyes had >2 line improvement in visual acuity

- The patient belonging to grade VI chemical injury in the study, didnot show any improvement in visual acuity in 1st week

- At the end of one week post-operative period, 80% eyes had improvement in limbal vascularisation of < or= 6 clock hours, 20% eyes had improvement >6 clock hours.

- 69.3% of eyes with grade IV injury did not have residual limbal ischemia at the end of one week, whereas the eye with grade VI injury the residual limbal ischemia was 6 clock hours.

- At the end of one month, in eyes with grade IV injuries, improvement in visual acuity = or>3 lines was seen in 61.5%.

- The results of improvement in visual acuity at one month were similar to the results at one week.

- The patient who presented with grade VI injury had a residual ischemia of 4 clock hours with symblepharon formation.

- All patients of grade IV and V chemical injury had no residual limbal ischemia or ocular surface abnormality at the end of one month.

CONCLUSION

CONCLUSION

Chemical injuries are one of the devastating cause of permanent visual loss in the young working population. By producing limbal ischemia and limbal stem cell deficiency, the chance of corneoscleral ulceration is common. It requires immediate and prompt medical and appropriate surgical intervention to prevent the permanent complications.

Our study shows that surgical intervention by tenoplasty in severe chemical injuries, improves the limbal ischemia and prevents corneoscleral ulceration and gives us a chance for future optical keratoplasty for visual rehabilitation.

PART III

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BIBLIOGRAPHY

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PROFORMA

PROFORMA

S.NO.

NAME

AGE

SEX

LATERALITY

MODE OF INJURY

CHEMICAL

CLINICAL FEATURES

1. VISUAL ACUITY
2. LIMBAL ISCHEMIA
3. CONJUNCTIVAL CHEMOSIS
4. GRADE OF INJURY BY DUA CLASSIFICATION
5. CORNEAL EPITHELIAL DEFECT
6. INTRAOCULAR PRESSURE
7. FUNDUS EXAMINATION
8. BLOOD PRESSURE
9. SERUM GLUCOSE LEVELS AND

10. URINE- ALB/SUGAR.

TIME OF TENOPLASTY

1ST WEEK POST-OP

1. VISUAL ACUITY
2. LIMBAL ISCHEMIA
3. CORNEAL EPITHELIAL DEFECT
4. SYMBLEPHARON
5. LID ABNORMALITIES
6. INTRAOCULAR PRESSURE

1ST MONTH POST-OP

1. VISUAL ACUITY
2. LIMBAL ISCHEMIA
3. CORNEAL EPITHELIAL DEFECT
4. SYMBLEPHARON
5. LID ABNORMALITIES
6. INTRAOCULAR PRESSURE

KEY TO MASTER CHART

M – Male

F – Female

RE – Right eye

LE – Left eye

BE – Both eyes

Limbal ischemia – in clock hours

Conjunctival chemosis – in %

PL – Perception of light

HM – Hand movements

S – Symblepharon

S.NO.	NAME	AGE	SEX	LATERALITY	MODE OF INJURY	CHEMICAL	VISUAL ACUITY	LIMBAL ISCHEMIA	CONJUNCTIVAL CHEMOSIS	GRADE OF INJURY	CORNEAL EPITHELIAL DEFECT	TIME OF TENOPLASTY	1ST WK VISUAL ACUITY	1ST WK LIMBAL ISCHEMIA	EPITHELIAL DEFECT	1ST MTH VISUAL ACUITY	1ST MTH LIMBAL ISCHEMIA	1ST MTH CORNEAL STATUS
1	Kalaicharan	20	M	RE	industrial	acid	6/18	6	50	4	P	3	6/12	No	No	6/6	No	No
2	Seetha	40	F	LE	Assault	acid	PL	12	100	6	P	7	PL	6	P	HM	4	s
3	Poornima	5	F	RE	Accidental	Alkali	6/60	7	60	4	P	7	6/36	2	p	6/12	No	No
4	Jaswant	3	M	RE	Accidental	Alkali		9	80	5	P	7		3	P		No	No
5	Joseph	43	M	LE	Accidental	acid	HM	6	60	4	P	6	HM	No	No	HM	No	No
6	Selvi	38	F	LE	Assault	acid	HM	10	100	5	P	3	HM	2	p	HM	No	No
7	Selvi	38	F	RE	Assault	acid	6/60	8	90	4	P	5	6/36	No	No	6/36	No	No
8	Baskar	27	M	LE	Accidental	Alkali	6/24	6	50	4	P	3	6/18	No	No	6/12	No	No
9	Baskar	27	M	RE	Accidental	Alkali	6/18	6	50	4	P	5	6/12	No	No	6/9	No	No
10	Selvaraj	20	M	RE	Accidental	acid	1/60	8	90	5	P	3	5/60	2	P	6/36	No	No
11	Selvaraj	20	M	LE	Accidental	acid	3/60	6	70	4	P	5	6/60	No	NO	6/18	No	No
12	Ravi	42	M	LE	Assault	acid	HM	9	90	5	P	3	2/60	2	No	5/60	No	No
13	Ravi	42	M	RE	Assault	acid	2/60	7	80	4	P	5	3/60	No	No	5/60	No	No
14	Janardhanan	37	M	RE	Assault	Alkali	PL	10	90	5	P	1	1/60	4	P	2/60	No	No
15	Rama	30	F	RE	Accidental	acid	6/60	6	80	4	P	2	6/60	No	No	6/36	No	No
16	meena	20	F	LE	industrial	acid	6/36	6	50	4	P	3	6/12	No	No	6/6	No	No

17	Jothi	38	F	LE	Accidental	acid	HM	8	90	5	P	6	HM	4	P	HM	No	No
18	Jothi	38	F	RE	Accidental	acid	3/60	6	60	4	p	8	6/60	2	No	6/60	No	No
19	Pushpa	16	F	RE	Accidental	Alkali	5/60	7	80	4	P	3	6/60	2	p	6/18	No	No
20	Pushpa	16	F	LE	Accidental	Alkali	6/60	6	60	4	P	7	6/36	2	p	6/12	No	No